From the American Venous Forum

Definitive diagnosis and definitive treatment in chronic venous disease: A concept whose time has come

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It has been a great honor for me to serve as President of the American Venous Forum (AVF) during this past year, and I wish to thank the members of the society for giving me this wonderful experience. It is exciting to be associated with the AVF in its formative years, when opportunity abounds to contribute to the directions that the society will take as it pursues its purpose of improving the care of the venous patient through research, education, and clinical investigation of venous diseases.

The stimulating opportunity to present a presidential address leads to global thoughts about the society and the specialty it represents. As one looks at the field of venous disease, there is a sense of an orphan specialty in search of a home. Venous disease involves many facets of medicine, but there is no single specialty that has adopted overall responsibility for it. Certainly it is a big enough field to warrant attention, because it affects as much as 50% of the adult population in one form or another.[1] Clearly it is a serious enough problem, with its acute manifestation of deep venous thrombosis (DVT) and pulmonary embolism representing a major cause of death,[2] and its chronic form a leading cause of work disability.

But the fact is that no specialty has taken full responsibility to study and "conquer" the problems of venous disease in the way that other major disease entities have been assumed by specialty areas, and there have not been any great teaching institutions that have earned a dominant reputation for managing venous disease in America. This leaves a huge void that yearns to be filled. In my mind, the AVF is well constituted to become a major factor in filling this void.

The AVF has a high representation of academia in its ranks, which gives rise to a strong accent on clinical and basic research in its proceedings. Because the impact that an organization can produce is shaped by the individuals who make up the organization, I believe that the great contribution to be made by the AVF in the future will result from its ability to define basic principles of diagnosis and treatment because of its research and teaching capability. A recent example of this ability was Dr. Hobson's presidential address[3] to this society 1 year ago, which was devoted to the future of research in venous disease.

As we move forward, it is clear that there is a need to organize the findings we accept as established "facts" in an orderly manner and to adopt a common language with definitions of terms to facilitate meaningful communication. This effort requires that we reexamine those beliefs that were so deeply ingrained in us by our teachers that were based on clinical impression but were not tested in the objective realm by reproducible methods. Over the years, pure clinical diagnosis in venous disease has been found to be inaccurate. This is true of many facets of venous disease, such as the clinical diagnosis of DVT, which has been proven unreliable; the clinical diagnosis of pulmonary embolism, which we recognize to be unreliable; and the clinical diagnosis of the postphlebitic leg, which is also unreliable.

If it can be accepted that the veins function optimally when they are both patent and competent, then any other condition is a compromise with normal function and needs to be controlled by the compen-

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sating capacity of the veins. It follows that the ideal of treatment is to restore both patency and competence to the diseased veins. When that restoration is not possible, partial correction will often result in a compensated state in which the venous drainage is restored sufficiently to allow the individual a normal way of life. The challenge is to identify the precise abnormalities that are present in the veins of patients who have chronic venous disease and to develop ways to either correct the abnormalities themselves or achieve a compensated state by ligation, stripping, or bypass that will improve patency and competence even if it cannot be totally corrected.

Until the advent of the color-flow Doppler scan, and the even more explicit duplex scan, diagnosis in venous disease was dominated by superficial inspection and professorial opinion that was based on experience without objective testing. The fallacy of this approach was forcefully made in acute venous disease by Dr. Jack Cranley and associates,[4] who pointed out that the clinical diagnosis of DVT is wrong half of the time and that the errors occur both in false-positive and false-negative diagnoses. This realization gave purpose to the requirement for objective diagnoses to justify treatment in DVT. My thesis is that these same principles need to be applied to chronic venous disease, in which clinical diagnoses can be equally deceiving.

PRESENT STATUS IN CHRONIC VENOUS DISEASE

In today's practice, the diagnosis of the patient who has chronic venous disease (CVD) is essentially random from doctor to doctor and from institution to institution. The completeness of the workup and the choice of test used to establish a diagnosis in CVD vary widely. The techniques of performing the venous tests and the quality control of the testing vary tremendously from laboratory to laboratory. Imprecise terms such as "chronic venous insufficiency" and "postphlebitic syndrome" are commonly used as definitive diagnoses without delineation of the physiologic or anatomic problem. Many, if not most, of the reports in the literature that deal with venous ulcer fail to define the clinical state in reproducible terms, so the reader is not informed whether the ulcer is due to disease in the superficial, deep, or perforator veins, whether the venous problem is one of reflux or of obstruction, or whether the cause of the problem is postthrombotic or primary venous disease. With this amount of imprecision, it should come as no surprise that confusion over results of treatment and of diagnosis is rampant in CVD.

The challenge now is to convert the present state in which a few "facts" are mixed with a lot of supposition into a new state in which basic facts are established by scientific study that can be reliably reproduced and formulated into a set of guiding principles for CVD. To do this we will need prospective studies of carefully defined clinical problems managed by alternative treatment techniques. These treatments need to be monitored by objective testing for a long enough period to establish their validity. This process has begun and will occur slowly and incompletely over time, but it can be facilitated by consciously defining our goals.

In my estimation there are several steps that must be in place for good clinical research to occur:

1. Define which patients are being treated. The process begins with precise diagnosis. The venous system is complex in its normal anatomy and physiology, and becomes far more complex in disease states. These complexities need to be identified by thorough pretreatment diagnosis achieved by objective, reproducible testing procedures. The new CEAP (clinical, etiologic, anatomic, pathophysiologic) classification of the AVF provides a start in this direction.[5]

2. Define which tests to use when. There need to be recommendations of minimum testing to justify a given diagnosis. The specificity and the accuracy of various tests for specific pathologic problems remains to be defined. Because the variability of venous testing is immense from one institution to another, quality control standards are necessary for vascular laboratories.

3. Prospective studies. Armed with thorough diagnoses that are reproducible from institution to institution, and using testing methods of comparable reliability from institution to institution, prospective interinstitutional case series can be designed with adequate numbers of cases to produce conclusive results in a reasonable period of time. These series can compare surgical and medical variations in the management of comparable problems (apples to apples). In this way, definitive diagnoses can be used to study variations in definitive treatment alternatives.

4. Adequate follow-up. CVD, like a glacier, moves slowly but forcefully over time. There are dynamic changes that demand long-term follow-up if correct conclusions are to be reached. Two-year results are likely to be misleading. Five-year results may be a minimum time. Eight- to 10-year results are about as long as follow-up can be practical because patients move away, change lifestyles, develop new illnesses, die, or become too disinterested. In addition to simple follow-up in CVD, there needs to be a

comparison of the preoperative and postoperative activity level of the patient before it can be concluded that a treatment has been successful.

A NEW BEGINNING

The CEAP classification. With the critical problem of confused communication with regard to CVD in mind, an international panel of experts gathered in 1994 under the auspices of the AVF to develop a consensus statement about the diagnosis and classification of CVD.[6] The result was the new CEAP method of classification, in which the elements of a correct diagnosis in CVD were determined to be the clinical condition, the etiologic mechanism of the condition, the anatomic distribution of the problem, and the pathophysiologic mechanism of the development of the problem.

These categories were developed into an acronym much as we use to describe malignant tumors, in which each main category has subcategories and a shorthand method is used to express the condition. In the CEAP classification there are six subheadings under Clinical condition, three under Etiologic mechanism, three main headings under Anatomy that are subdividable into eighteen segments, and three headings under Pathophysiology. A cryptic method of expressing a thorough diagnosis emerges. For example, "C2,3-a-Ep-As-Pr2,3" describes a case of varicose veins with swelling, free of pain; caused by primary venous disease; limited to the superficial veins; having reflux in the greater saphenous vein of the thigh and calf as the mechanism of development. "C2,4,6-s-Es-As,d,p-Pr2,3,14,15,18-o13" describes a case of varicose veins with stasis skin changes and ulceration, accompanied by pain; caused by postthrombotic disease; affecting the saphenous, deep, and perforator veins; causing reflux in the entire greater saphenous vein and in the popliteal and crural veins and in the calf perforator veins, and obstruction in the superficial femoral vein.

This clearly is a new standard of diagnosis in CVD that will require a learning curve for all of us to master. The consensus committee wrestled with the dilemma of a complicated but thorough definitive diagnosis versus the need to keep it short and simple enough to be practical. It concluded that anything less than its present form would be incomplete and would negate the effort to achieve reproducible diagnoses in CVD. In adopting this format, the committee recognized that CVD is complex and cannot be oversimplified if it is to be understood. For too long we have underestimated and underdiagnosed chronic venous problems, with the result that our understanding of them remains incomplete up to the present time. The elements of the CEAP classification are analogous to the elements customarily included in the diagnosis of arterial problems in which we determine the cause, the anatomic distribution, and the pathophysiologic mechanism in each clinical condition (Table I).

Table I. Elements of vascular diagnoses—classification

Arterial	*Venous*	
foot ulcer	leg ulcer	C
atherosclerosis vs embolic	primary vs postthrombotic	E
femoral-popliteal	deep (fem-pop-tib)	A
stenosis/occlusion	reflux/obstruction	P

The ad hoc committee went further and, in an addendum to the actual classification, provided suggestions for judging the severity of the venous insufficiency by grading the signs and symptoms of the disease process. It also described the specific applications of the various tests used in CVD.[5]

Publication of the document has been carried out around the world during the past 18 months. It has been officially adopted in the United States by The American Venous Forum and the North American Society of Phlebology, approved by the joint council of the Society for Vascular Surgery/International Society for Cardiovascular Surgery, North American Chapter, and published in the Update of the Reporting Standards in Venous Disease as the new standard for reporting in CVD,[7] and has been published in the dermatologic literature.[8] It has or soon will appear in vascular and phlebologic journals in the United States and around the world, including Japan, Australia, South America, and multiple journals throughout Europe including England, France, Germany, Austria, and Italy. It has been translated into French, German, Italian, Japanese, and Spanish. Worldwide acceptance of this single classification is necessary to ensure proper communication internationally.

Many problems will surface with implementation of the CEAP method, and these problems will need to be processed over time. But it is a beginning. The experience of having an international group reach consensus speaks to the marvelous ability of Dr. Andrew Nicolaides to conduct a consensus conference and represents a pinnacle of cooperation among clinicians and scientists around the world to come together and embrace a solution to a common problem.

Personal experience with CEAP. To evaluate the CEAP classification and test the value of highly specific diagnoses, we have examined two series of

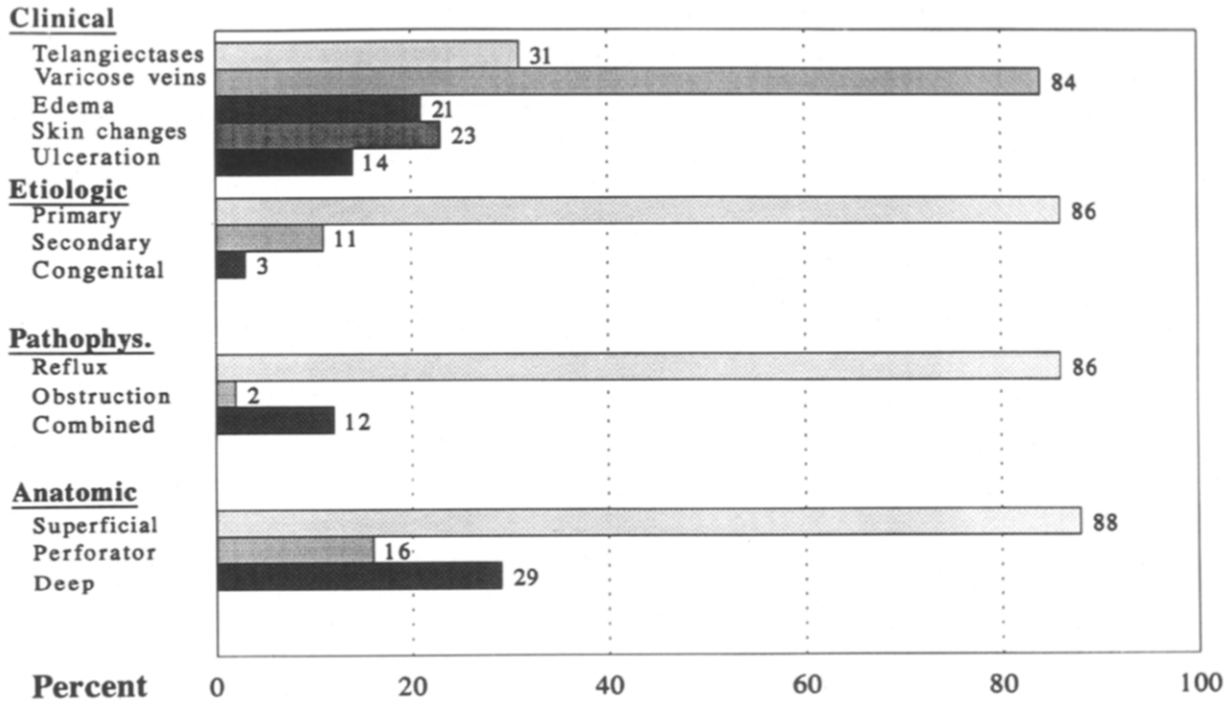

Fig. 1. Series I. CEAP classification of 102 consecutive cases of CVD. (Reprinted from Kistner RL, Eklof B, Masuda EM. Mayo Clin Proc 1996;71:338-45.)

patients. Series I was a consecutive series of 102 patients who had any complaint of chronic venous insufficiency. Series II was a consecutive series of 56 venous ulcers.

Each patient was evaluated with a history, a physical examination, and a color-flow Doppler scan performed by an experienced vascular surgeon. Additional tests began with duplex scanning for all patients who had clinical findings beyond the level of telangiectasia and branch varicose veins. Plethysmography (using air plethysmography in most cases), venous pressure, and ascending and descending phlebography were applied selectively to define aspects of the diagnosis in more complicated problems.

The results of these two series are presented in Figs. 1 and 2.[9] Points of particular interest included a great predominance of primary disease in both series I (86%) of consecutive CVD cases and in series II (71%) limited to the ulcer population. There was also an 8-to-9:1 predominance of reflux disease over obstructive disease in both groups; this finding is consistent with the literature and is demonstrative of the dominating clinical importance of axial reflux in CVD.

In series I (consecutive chronic venous cases) there was a high proportion of minor problems presenting as telangiectases and minor varicose veins, some of which did not involve the saphenous vein itself. These problems are managed with a minimum of diagnosis and treated with sclerotherapy; they demonstrate the huge numbers of these problems in the population. In spite of this, 23% of the series had skin changes and 14% had ulcer problems. These percentages would vary widely from series to series, but the knowledge of their distribution is of value for any practice in its own self-analysis.

In the breakdown analysis of anatomic involvement in the ulcer series, reflux was limited to the saphenous veins in 29% of cases that were caused by primary disease and in none of the cases that were caused by postthrombotic disease; this finding identifies a marked difference between the two causes. These superficial primary cases are a specific group of patients whose ulcers are amenable to cure by simple saphenous vein surgery. Those ulcers in the primary cases with saphenous plus perforator incompetence and a competent deep system by duplex study (32%) are also amenable to excellent results by relatively simple outpatient surgery.[10]

The finding that primary disease was the cause in 70% of ulcer cases in series II supports the need for definitive diagnoses to include the cause of the condition before comparing results between series. If the series had 70% of ulcers caused by postthrombotic

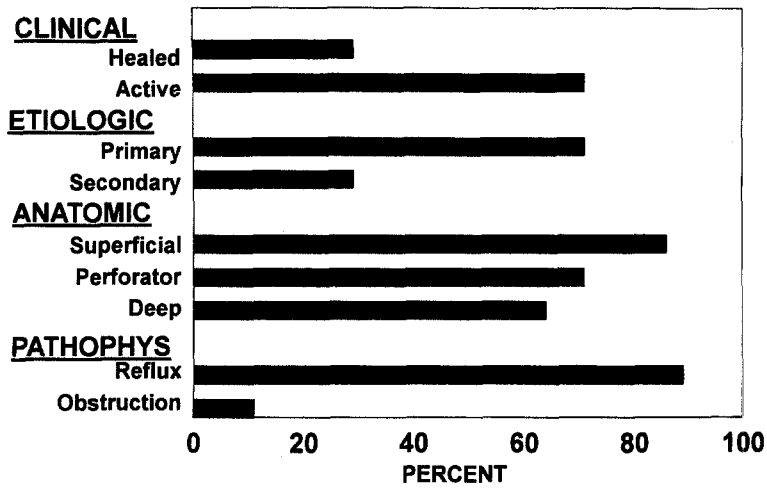

Fig. 2. Series II. CEAP classification in 56 cases of venous ulceration.

disease, any treatment method might have quite different results over the long term.

The ulcer cases with secondary (postthrombotic) cause were different from the primary cases in that every secondary case showed deep vein disease, as opposed to 50% with deep vein disease in the primary cases. In addition, the saphenous vein (75%) and the perforators (81%) were usually involved. This finding speaks to the presence of widespread venous derangement in those postthrombotic cases that develop ulceration. In our experience, postthrombotic cases develop higher recurrence rates after deep vein reconstruction than do primary cases if observed long enough (at least 4 to 6 years)[11] (Fig. 3), and we attribute this to the supposition that the venous derangement is greater in these extensive postthrombotic patients than it is in most of the primary cases.

There are important differences between primary and secondary venous disease. The morphologic difference between the extensive intraluminal venous destruction found in the postthrombotic vein and the normal-appearing intima of the primary refluxing vein is dramatic. Because the clinical symptoms may be identical while the pathologic changes are vastly different, the clinical diagnosis is not a good predictor of the extent of venous damage. Physiologically, primary disease produces pure reflux, whereas postthrombotic disease may cause pure reflux, nearly pure obstruction, or mixed reflux and obstructive changes. The difference between primary and secondary also extends to treatment where the long-term results of reconstruction for primary reflux are better than those of postthrombotic reflux (Fig. 3) in most of the published series.[12-15]

A striking finding that emerged from analysis of the anatomic distribution in the ulcer series was that axial reflux in both thigh and calf was present in 51 of 53 cases (96%). Of the two exceptions, there was isolated perforator disease in one case, and in the other case the reflux was isolated to the popliteal-tibial plus the perforator veins. Both of these cases were of primary cause.

The conclusion from these observations is that the CEAP classification, using objective testing appropriate to the severity of the clinical problem, has practical results in sorting out patients for surgery and other treatment. At the same time, it affords the opportunity to interrelate the clinical, anatomic, etiologic, and pathophysiologic phenomena of CVD to each other.

INFLUENCE OF SPECIFIC DIAGNOSIS ON TREATMENT

If the concept of a thorough diagnosis that requires specific testing is to be accepted by the public, it will be important to demonstrate that the additional testing pays off by effecting an improvement in the management of the patient. At present, physicians are in a quandary about how to treat patients who have serious chronic venous states, such as advanced skin changes and ulceration. An example is the comparison of the management of venous ulcer patients by medical and by surgical methods according to today's literature. To date, it is inappropriate to compare the results of published series that were treated medically with those treated surgically because the medical cases are not thoroughly diagnosed and do not represent a uniform population, whereas the patients in the reconstructive surgical group have all undergone thorough workups to plan surgery, and they do represent a relatively uniform population. The medi-

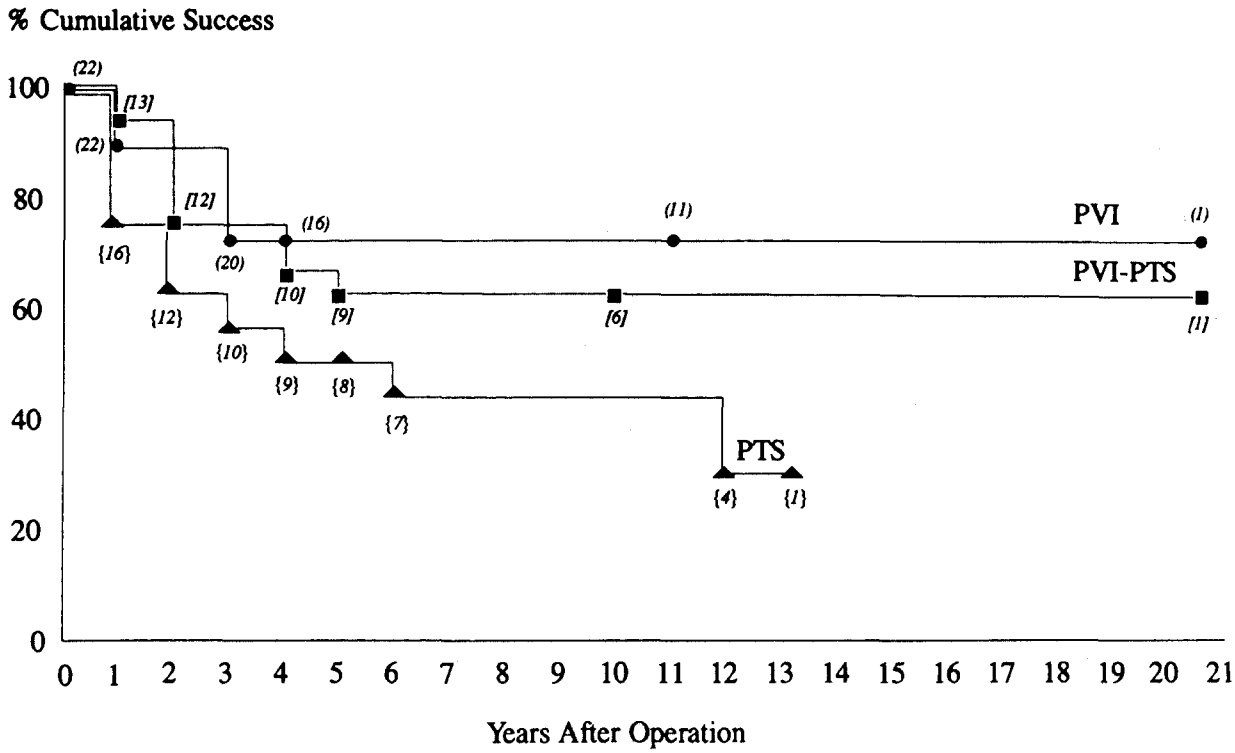

Fig. 3. Life table of results of deep venous reconstruction in cases of primary, secondary, and mixed causes. (Reprinted from Masuda EM, Kistner RL. J Vasc Surg 1994;19:391-403.)

cal series may consist of mixtures of primary and secondary reflux; mixtures of reflux and obstruction; some with proximal disease, others with distal disease, and some with both. Even the clinical status may be quite different because the reconstructive surgical cases by and large represent patients who have recurrent difficult problems that failed to respond to simpler treatment methods, whereas the medical cases vary from first-time clinical cases to end-stage non–surgically repairable venous insufficiency.

I have a favorite slide that is appropriate to this point. It shows six extremities in different patients in all of whom the findings would prompt the term "postphlebitic leg," but in whom definitive testing revealed six different diagnoses, and only one is a postthrombotic case. The others were one case each of ulceration caused by simple saphenous vein reflux, ulceration caused by isolated perforator reflux, ulceration as a result of combined saphenous and perforator incompetence, and advanced skin changes and a healed ulcer as a result of extensive primary reflux disease affecting saphenous, perforator, and deep veins. There was one case of 4+ skin changes and deep discoloration caused by repeated trauma in a diabetic patient whose veins were normal. Of these extremities, four were a result of strictly primary venous reflux disease, one had no venous disease, and only one had postthrombotic disease. The external appearance of "postphlebitic syndrome" would have been in error in five of the six cases.

The most important aspect of these six extremities are the implications for treatment: the one with no venous disease needs no treatment; the three with superficial and perforator primary reflux disease can be treated with outpatient surgery and can expect to be cured because the deep systems are normal; the one with extensive primary disease in saphenous, perforator, and deep veins requires saphenous and perforator surgery and may require deep vein valvuloplasty depending on the response; and the one with extensive postthrombotic changes would also be surgically managed by obliteration of the saphenous and perforator veins, but the deep reconstruction, if needed, would be achieved by a valve substitution rather than a valve repair method.

In analysis of the CEAP experience from series I and series II, at least four points stand out.

First, in ulcer cases whose cause is primary disease, one fourth were caused by pure saphenous reflux and another one fourth were a result of combined saphenous and perforator reflux in the presence of a normal deep system. These patients are readily treatable by

performing outpatient surgery on the saphenous and perforator veins and can be expected to have a very high rate of long-term cure.[16] This experience is not unique, having been reported in other series,[17] but the important thing is that these patients represent a large group of ulcer cases that are amenable to relatively simple surgery, and many of these patients can expect to be free of the need to wear support stockings indefinitely.

There is a common belief that all patients who have a venous ulcer will need to wear elastic stockings for the rest of their lives, regardless of the treatment method used. This is just not true. Vein stripping for patients who have reflux limited to the saphenous, and vein stripping plus perforator interruption for those who have combined primary reflux limited to saphenous and perforator veins, will be permanently curative with relief of need for elastic support in >80% or more of cases.[10] This alone constitutes adequate reason for an accurate workup. Add to these our long-term follow-up of patients who have advanced primary deep vein disease treated surgically by valvuloplasty, in whom 73% were free of recurrent ulceration after 10 years, and in whom 30% stopped wearing elastic support[11] and were free of recurrence. When all of these cases are accounted, up to half of all patients who have an ulcer can be relieved of elastic support after selective surgery.

Second, all of the patients who had ulcers as a result of postthrombotic disease had involvement of the deep veins. The pathophysiologic mechanism of this deep vein involvement usually consists of a combination of reflux and obstruction, with one or the other being physiologically dominant. In contrast, only half of the ulcer cases caused by primary disease had deep vein involvement, and this was invariably pure reflux. This difference accounts for a much better prognosis for surgical therapy in primary disease than in secondary disease.

Third, of the cases that had venous ulceration, 96% were found to have reflux or obstruction in both the thigh and the calf. Although this finding is consistent with the importance of the popliteal valve as the gatekeeper of the deep veins of the lower leg,[18] it emphasizes the importance of proximal reflux in cases with advanced signs of venous insufficiency.[19] The reflux that causes ulceration can be localized to just the superficial veins or can involve superficial and deep veins; cases with only deep vein reflux appear to have pain but are not likely to develop ulcers or skin changes.

Fourth, reflux dominates over obstruction as a cause of ulceration and of advanced skin changes. It is the sole pathophysiologic finding in the primary cases, and it occurs together with obstruction in the postthrombotic cases. In the latter case, either reflux or obstruction may dominate physiologically.

The importance of specific and complete diagnoses extends to the medically treated as well as the surgically treated CVD cases. In the medical cases, there are choices of therapy and questions of medications and dressings that require investigation. At this point, we do not know if the site of the disease or the response of an obstructed versus a refluxing venous system makes a difference in nonsurgical treatment results. It is likely, but not proven, that the long-term clinical behavior of an extremity that is symptomatic on the basis of primary reflux in the saphenous vein will behave better than a similar degree of postthrombotic reflux mixed with obstruction that affects the deep and the saphenous veins.

PERSPECTIVE

The CEAP classification, as important as it is, only represents a beginning. It remains to define the testing process more precisely. The limits of reliability of a given test in a given pathologic state need to be agreed on and published, and the reliability of these tests needs to be standardized in each laboratory to provide interinstitutional parity and lead to reproducible results in clinical practice. A consensus conference on this subject is to be held later this year in Europe under the leadership of Dr. Nicolaides and the International Union of Angiology and will include the participation of AVF members. This is a necessary step in building toward interinstitutional studies and reproducible results.

Until the use of the CEAP classification for completeness of diagnosis and the employment of reliable tests to accurately identify pathologic problems are in place, we will not be able to conduct the large-scale clinical studies with the statistical power afforded by larger numbers that will lead to sustainable conclusions about the relative value of various treatment methods in clinical practice.

CONCLUSION

The field of CVD has been awaiting the advent of definitive, economical, and noninvasive safe testing methods. Now that we have these methods, we appear to be on the cusp of an explosion of scientific development in CVD. I believe a whole new attitude about venous disease will be required to progress from our present state where science is mingled with fiction to a new state of well-grounded, objectively tested facts in CVD that will stand the test of time. This

attitude will accept that CVD is complex and important, and as such it requires definitive diagnoses based on objective criteria. In current lingo, this represents a paradigm shift.

It is true that we only treat what we suspect to be present through the diagnostic evaluation of any patient. The importance of a complete diagnosis that meets the CEAP criteria lies in the information it affords for understanding of the disease process and selection of specific treatment for individual problems. This facilitates choosing medical and surgical approaches and allows selection of surgical candidates for specific surgical procedures. It provides the basis for longitudinal and interinstitutional studies of alternative treatment techniques. This discipline over time will lead to new imaginative approaches to correct abnormalities of obstruction and incompetence. It is for these reasons that I believe specific diagnosis and specific treatment in CVD is a concept whose time has now come and that this will lead to the dawning of a new level of scientific pursuit in CVD.

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Important Notice

Effective October 1, 1996, all new manuscript submissions should be sent to the new editorial office (Journal of Vascular Surgery, Editorial Office, Toronto Hospital, Eaton 5-312, Toronto, Ontario, Canada, M5G 2C4) to the attention of K. Wayne Johnston, MD, and Robert B. Rutherford, MD. Manuscripts received before October 1, 1996, and those currently in the process of review will remain the responsibility of Editors Calvin B. Ernst, MD, and James C. Stanley, MD.